10.0 CHILDHOOD BRAIN CANCER

STATEMENT TO THE PUBLIC

The reviewers expressed their judgments using two distinct sets of guidelines to evaluate the evidence:

- ***Using the traditional guidelines of the International Agency for Research on Cancer (IARC) for childhood brain cancer, their classifications for EMFs was "inadequate" (IARC's Group 3). Panels convened by IARC and the National Institutes for Environmental Health Sciences also thought the evidence was "inadequate" to make a classification.***

- ***Using the Guidelines developed especially for the California EMF program, two of the reviewers were "prone to believe" that high residential EMFs do NOT cause any degree of increased risk of childhood brain cancer, one "close to the dividing line between believing or not believing" in any effect.***

The EMF Program's policy analysis required each of the three DHS scientists to express in numbers their individual professional judgments that the added personal risk suggested by the epidemiological studies was "real." They did this as a numerical "degree of certainty" on a scale of 0 to 100. The three scientists each came up with a graph that depicts their best judgments with a little "x" and the margin of uncertainty with a shaded bar: The differences in certainty between the three reviewers arises primarily from how sure they were that they could rule out study flaws or other explanatory agents and how much the evidence on one disease influenced certainty in the findings for other diseases.

CONDITION	REVIEWER	IARC CLASS	CERTAINTY PHRASE	DEGREE OF CERTAINTY IN CAUSALITY FOR POLICY ANALYSIS
Childhood Brain Cancer				0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
	1	Inad. 3	Close to Dividing Line	
	2	Inad. 3	Prone Not to Believe	
	3	Inad. 3	Prone Not to Believe	

10.1 EPIDEMIOLOGICAL EVIDENCE REGARDING CHILDHOOD BRAIN CANCER

Figure 10.1.1 Studies Relating Childhood Brain Cancer to Proximity to Power Lines and Prenatal Exposure to Electric Blankets

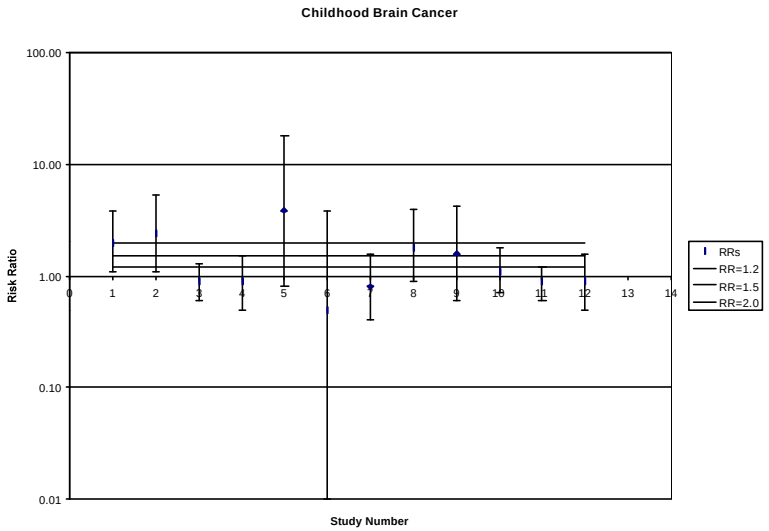

TABLE 10.1.1 KEY TO FIGURE 10.1.1

STUDY	NO.	INDIVIDUAL ODDS RATIO	LOWER CL	UPPER CL	
(Savitz et al., 1988)	1	2.00	1.10	3.80	OHCC
(Wertheimer & Leeper, 1979)	2	2.40	1.08	5.36	OHCC
(Preston-Martin et al., 1996b)	3	0.90	0.60	1.30	OHCC
(Gurney et al., 1996)	4	0.90	0.50	1.50	OHCC
(Tomenius, 1986)	5	3.90	0.80	18.00	<150 m from line
(Feychting & Ahlbom, 1993)	6	0.50	0.01	3.80	<50 m from lines
(Tynes & Haldorsen, 1997)	7	0.80	0.40	1.60	<50 m
(Savitz, John & Kleckner, 1990)	8	1.80	0.90	4.00	Electric Blanket
(Kuijten, Bunin & Nass, 1990)	9	1.60	0.60	4.20	Electric Blanket
(McCredie, 1994)	10	1.10	0.70	1.80	Electric Blanket
(Preston-Martin et al., 1996)	11	0.90	0.60	1.20	Electric Blanket
(Gurney et al., 1996)	12	0.90	0.50	1.60	Electric Blanket

Figure 10.1.2 Studies of Childhood Brain Cancer and Measured Magnetic Residential Fields

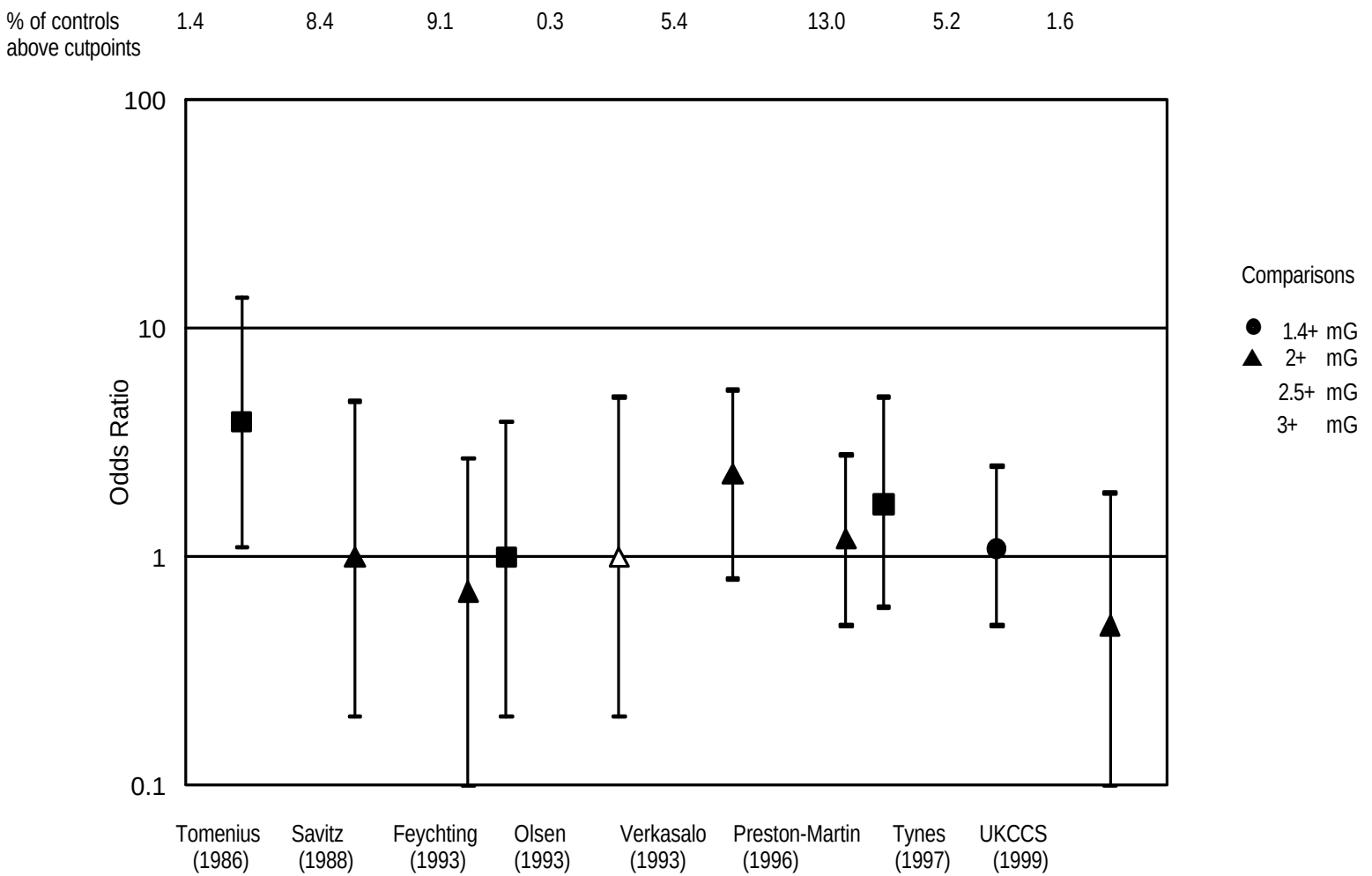

TABLE 10.1.2

Investigator, Date	Design	Definition of Case Series[1]	Age Group	Number of Cases/ Control or Cohort	Control Selection Procedure	EMF Exposure Surrogate[2]				
						1	2	3	4	5
(Wertheimer & Leeper, 1979)	Case-control	CNS	0-18	66/66	Birth Records	X[3]				
(Savitz et al., 1988)	Case-control	brain	0-14	59/259	RDD	X		X[4]		X
(Tomenius, 1986)	Case-control	CNS	0-18	294/253	Birth Records		X	X		
(Feychting & Ahlbom, 1993)	Nested Case-control	CNS	0-15	33/141	Cohort		X	X	X	
(Olsen et al., 1993)	Case-control	CNS	0-14	624/1872	Population Register		X		X	
(Verkasalo et al., 1993)	Cohort	CNS	0-19	39/134, 800	-----				X	
(UKCSS, 1999)	Case-control	CNS	0-14	359/371	Population Register		X	X	X	
(McCredie, 1994)	Case-control	CNS	0-14	82/162	Electoral Role					X
(Gurney et al., 1996)	Case-control	brain	0-19	133/270	RDD	X				X
(Preston-Martin et al., 1996b)	Case-control	brain	0-19	298/298	RDD	X		X		X
(Kuijten et al., 1990)	Case-control	astrocytoma	0-15	163/163 matched pairs	RDD					X
(Tynes & Haldorsen, 1997)	Nested Case-control	CNS	0-14	156/639	Cohort		X		X	

From Kheifets et al., 1999

[1] All studies (except for Wertheimer-Leeper) are based on incident cases.
[2] Exposure surrogate: (1) wire code, (2) distance, (3) measured fields, (4) calculated fields, (5) appliance use.
[3] HCC/LCC comparison only.
[4] Spot measurements only.

Figure 10.1 and its key show associations between exposure ("wire code," distance from lines, and appliance use), and childhood brain cancer. With regard to the first seven studies in the graph, which examined distance from power lines and wire code, 3 showed ORs >1.00 (exact binomial probability = 0.27). Of 5 studies reporting associations with prenatal electric blanket exposure, 3 had ORs > 1.0 (p = 0.31). For the most part, the studies had wide confidence intervals.

Figure 10.2 shows eight studies reporting associations with measured magnetic fields four reported RR > 1 (p = 0.27). Once again the confidence limits around the odds ratios are wide.

10.2 Arguments For and Against Causality

Table 10.2.1

CHANCE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The larger and better designed studies show no statistically significant results.	(F1) The power of these studies may be insufficient to detect an effect of the rare higher exposures.	(C1) A meta-analysis by Wartenberg (Wartenberg, 1998) and an inspection of the associations above and below 1.00 for wire codes, measurements, and the history of appliance use all reveal a pattern which could be due to chance.
(A2) This pattern of results could be due to chance.		(C2) Several of the case control studies had several hundred incident cases accumulated over a number of years. Because childhood brain cancer is a rare condition, it will be difficult to conduct larger studies.

TABLE 10.2.2

BIAS		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Wertheimer (Wertheimer & Leeper, 1979) exposure assessment not done blindly could bias upward.	(F1) Wire codes were associated with leukemia in Los Angeles and Sweden. Wertheimer (Wertheimer & Leeper, 1979) blindly validated a sample of wire codes. There was no evidence for bias from lack of blinding.	(C1) The associations with childhood brain cancer are less consistent than is the case with leukemia and there is nothing about the study decisions which suggest biases operating in these studies that are not operating in leukemia studies.
(A2) Savitz (Savitz et al., 1988) had mobility criteria which produced selection bias and inflated the OR.	(F2) Poole (Poole, 1996) suggests mobility bias is not an explanation of the Savitz findings.	(C2) If the greater than 1.00 ORs from well-designed brain cancer studies are discarded as biased then their leukemia results should be discarded too. Yet those leukemia results are not inconsistent with results from later better designed leukemia results. The reviewers rely on chance, not bias, to explain the pattern of evidence.
(A3) High case fatality in the cases associated with high wire codes would falsely inflate wire code/brain cancer association in Wertheimer's (Wertheimer & Leeper, 1979) mortality study.	(F3) The Preston-Martin (Preston-Martin, 1989) study gathered controls concurrently after 1989. The control series matching cases before that time has a falsely low prevalence of underground lines, which biased the OR for underground lines upward. Preston-Martin cases also were lost to follow up. This may have biased the wire code association downward.	(C3) Imprecise exposure information may be pulling the associations toward the null.
(A4) The Preston-Martin (Preston-Martin et al., 1996b) cases and controls lost equal numbers of subjects to follow up. The null result is not a biased result as alleged in F3.	(F4) Wire codes for distribution lines do not work well outside of Denver hence the null results of wire code studies elsewhere.	
(A5) The Gurney (Gurney et al., 1996) study is good quality and its null result should pull down confidence.	(F5) Non-differential exposure misclassification biases associations toward the null for measurements, estimated historical fields, and wire codes.	

BIAS		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A6) Wire code for distribution lines can work elsewhere than Denver, contrary to the allegation in F4. While wire codes were developed for the Denver utility system, wire code associations with leukemia were seen in Los Angeles (London et al., 1991). The Preston-Martin study also was done in Los Angeles, and its null result cannot be discounted on the basis of poor wire codes.	(F6) The numbers available to study appliances are small, which leads to inconsistencies.	
(A7) Null results from wire code studies need to attract the same consideration as results with ORs greater than 1.00.	(F7) Not all appliances that patients might suspect and over-report are associated with disease, so there is little direct evidence of recall bias.	
(A8) Appliance studies are inconsistent and subject to recall bias.		

TABLE 10.2.3

CONFOUNDING		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The associations are inconsistent.	(F1) Controlling for known causes of childhood brain cancer made no difference in results.	(C1) The reviewers see no evidence that confounding explains the pattern of epidemiological evidence.
(A2) The only two statistically significant studies are from Denver. There may be confounding in that particular location.	(F2) Special confounding was invoked for the leukemia studies, too, and despite case-specular studies for neighborhood factors (Zaffanella & Hooper, 2000) and traffic (Pearson et al., 2000), no such confounder was found.	
(A3) The causes of childhood brain cancer are not understood, so one cannot control for these unknown confounders.	(F3) Why would confounding only occur in the studies with ORs greater than 1.00?	
	(F4) To invoke confounding, one needs specific evidence that it is present, not generic invocation. to dismiss association with which one disagrees.	

TABLE 10.2.4

STRENGTH OF ASSOCIATION (*LARGE ENOUGH TO BE CAUSE NOT BIAS?*)		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) The overall association is so close to 1.0 as to be vulnerable to bias and confounding and thus should be ignored. It is so close to 1.0 that it should be considered null in any case.	(F1) Not all the associations in all the studies are so small.	(C1) Taken as a whole, the evidence is not compatible with an effect that is much different than 1.0. Unspecified bias and confounding could easily occur, but chance is a more salient concern here.

TABLE 10.2.5

CONSISTENCY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) One should only consider statistically significant results.	(F1) One should look at all the evidence.	(C1) The pattern of associations is not consistent and there are no really strong associations.
(A2) Most of the studies show no statistically significant results.	(F2) It is not all null.	
(A3) About half the wire code and the minority of the measurement studies have ORs below 1.	(F3) Overall, it is compatible with an OR of 1.2 with wide confidence intervals.	
(A4) The appliance ORs are inconsistent and modest.		
(A5) This should pull down confidence a lot.		

TABLE 10.2.6

HOMOGENEITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Only the early, poor-quality Tomenius (Tomenius, 1986) paper showed a statistically significant association with measurements. Judging by Figure 10.2, the subsequent six studies did not achieve statistical significance for measurements or wire codes.	(F1) The associations are not all null. Something may be going on.	(C1) Even among the studies reporting RRs greater than 1.0, the pattern of odds ratios is heterogeneous. The later studies are less supportive.
(A2) Most of the wire code and appliance studies did not reach statistical significance.		
(A3) The studies are consistent in their lack of support.		
(A4) The later, better studies are less supportive.		

TABLE 10.2.7

DOSE RESPONSE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Within individual studies and between studies there is no orderly increase in risk as dose increases.	(F1) The number of children at the higher exposures is small enough that one's ability to discern dose-response relationships is not good.	(C1) The lack of power to detect dose-response relationships at the high end of residential exposures means that the lack of a dose-response relationship does not pull down confidence as much as the presence of a clear relationship would pull it up.
(A2) This should pull down confidence a lot.	(F2) Perhaps childhood brain cancer requires even higher exposures than childhood leukemia.	
	(F3) Imperfect exposure assessment can obscure dose response relationships.	

TABLE 10.2.8

COHERENCE/VISIBILITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Everyone is exposed to electricity so an epidemic should have been seen by now.	(F1) There has been an increase in childhood brain cancer (NCI, 1991).	(C1) If there is any observable effect, it would be from the rare high exposures and with a modest effect not easily detected in national rates.
		(C2) Brain cancer trends are affected by trends in diagnostic procedures.

TABLE 10.2.9

EXPERIMENTAL EVIDENCE		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Animal bioassays for brain tumors have been null.	(F1) One cannot always predict cancer type in humans from animal bioassays.	(C1) Null results in a non-sensitive test do not have as much weight as a positive result would have.
	(F2) Testing a few aspects of a complex mixture on the assumption that the risk increases monotonically into high doses with a non-human species is not a sensitive test for a complex mixture like EMFs.	
	(F3) Experiments at high doses on general bioeffects should increase confidence.	

TABLE 10.2.10

PLAUSIBILITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) There is no coherent mechanistic explanation based on agreed-upon experimental results on how exposure to residential EMFs could lead to physiological effects and then brain cancer.	(F1) Agents that cause harm often have no mechanistic explanation for a long time.	(C1) The lack of a mechanistic basis does not pull down confidence as much as the presence would pull it up.

TABLE 10.2.11

ANALOGY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
See generic discussion.		

TABLE 10.2.12

TEMPORALITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
See "Generic Issues" chapter.		

TABLE 10.2.13

SPECIFICITY		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
See "Generic Issues" chapter.		

TABLE 10.2.14

OTHER DISEASE ASSOCIATIONS		
AGAINST CAUSALITY	**FOR CAUSALITY**	**COMMENT AND SUMMARY**
(A1) Without mechanistic justification, other disease associations should have no bearing.	(F1) Associations with adult leukemia and brain cancer and childhood leukemia should boost confidence in the credibility of childhood brain cancer as caused by EMFs.	(C1) The other associations should have some weight.

TABLE 10.2.15

SUMMARY TABLE FOR CHILDHOOD BRAIN CANCER			
	HOW LIKELY IS THIS ATTRIBUTE OF THE EVIDENCE UNDER:		
ATTRIBUTE OF THE EVIDENC	**"NO-EFFECT" HYPOTHESIS**	**CAUSAL HYPOTHESIS**	**HOW MUCH AND IN WHAT DIRECTION DOES THIS ATTRIBUTE CHANGE CERTAINTY?**
Chance is credible explanation.	Likely		Chance has not been ruled out .
Upward bias not suggested for body of evidence.	Possible	Possible	None
Confounding unlikely.	Possible	Possible	None
Combined, chance, bias, confounding	Likely	Possible	Chance has not been ruled out
Strength of association doesn't exceed possible confounding or bias.	Possible	Less possible	No impact or slight decrease
Not consistently above the null.	Possible	Less possible	No impact or slight decrease
Homogeneity lacking between size of effects in few positive studies.	Possible	Less possible	No impact or slight decrease
Dose response not clear in studies.	Possible	Less possible	No impact or slight decrease
Coherence/Visibility: temporal trends would not reflect these near-null effects.	Possible	Possible	None
Experimental evidence for brain tumors is null.	Possible	Less possible	No impact or slight decrease
Plausibility: lack of strong mechanistic explanation.	Possible	Possible	None
Analogy.	Possible	Possible	None
Temporality.	NA	NA	None
Specificity: no specific subtype of tumor. Adult brain cancer shows some association.	Possible	More possible	None to slight increase

10.3 POSTERIOR (UPDATED) DEGREE OF CERTAINTY AND IARC CLASSIFICATION

10.3.1 STATEMENTS OF INDIVIDUAL REVIEWERS

Reviewer 1 (DelPizzo)

Degree of Certainty: The results are less consistent than those for childhood leukemia. Therefore, chance becomes a plausible explanation. However, the other arguments against causality are unconvincing, so that in this reviewer's opinion, the combined pattern of evidence is many more times likely to occur if the association is causal than if EMFs were really harmless. The posterior level of certainty on a scale from 0 to 100 is about 45 ("Close to the dividing line between believing and not believing"). For the purpose of decision analysis, a range between 30 and 60 should be used.

IARC classification: 3 (inadequate evidence).

Reviewer 2 (Neutra)

Degree of Certainty: The pattern of epidemiological evidence is quite likely under the no-effect hypothesis, particularly with the later better designed studies. The speculations about bias and confounding have not changed the assessment much and the lack of support from animal and mechanistic streams of evidence pulled the confidence down a little further. The adult brain cancer and leukemia associations pull confidence up, but only somewhat. The overall evidence leaves this reviewer's confidence of a causal effect of EMFs on childhood brain cancer about what it was to begin with but with a range that extends somewhat higher. This leaves a median posterior degree of certainty of about 11, falling into the "prone not to believe" category. For the purposes of the decision analysis, values ranging from 2 to 45 would be scientifically defensible.

*IARC Classification***:** The inconsistent epidemiology and the unsupportive animal and mechanistic information would classify the EMF/childhood brain cancer evidence as insufficient or "inadequate" to implicate EMF as a carcinogen and falls into Group 3.

Reviewer 3 (Lee)

Degree of Certainty: The evidence of the human studies lack power, even those well-designed studies, making them difficult to evaluate and do not rule out chance as a possibility. In both the wire code and measurement studies there are about an equal number of reported relative risks above 1.0 as there are below 1.0. Also, confounding and bias cannot be ruled out and there is a lack of a dose response as well as supporting animal studies. However, this reviewer's posterior is slightly increased over the prior on the basis of evidence of an EMF association found for childhood leukemia, and to a lesser extent adult brain cancer. Hence, this reviewer's posterior degree of certainty for purposes of the policy analysis falls within the "prone not to believe" category with a median posterior certainty of 20 and a range of 10 to 40.

IARC Classification: The human evidence is inconsistent where bias, confounding, and chance cannot be ruled out. The animal studies are less than sufficient or "inadequate" for EMF as a carcinogen even though there is support from positive findings associated with leukemia. The evidence would imply a Group 3 classification.

10.3.2 SUMMARY OF THE THREE REVIEWERS' CLASSIFICATIONS

CONDITION	REVIEWER	IARC CLASS	CERTAINTY PHRASE	DEGREE OF CERTAINTY IN CAUSALITY FOR POLICY ANALYSIS
Childhood Brain Cancer				0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
	1	Inad. 3	Close to dividing line	
	2	Inad. 3	Prone not to believe	
	3	Inad. 3	Prone not to believe	

10.4 POLICY RELATED SCIENTIFIC ISSUES

The following tables deal with evidence relevant to potentially bioactive aspects of the EMF mixture, the shape of dose response curves (if any), evidence for unequal vulnerability or exposure (if any), and the state of the science.

10.4.1 QUESTIONS RELEVANT TO DOSE RESPONSE AND POLICY

TABLE 10.4.1

HOW CONFIDENT ARE THE REVIEWERS THAT SPECIFIC EXPOSURE METRIC OR ASPECT OTHER THAN 60 HZ TWA MAGNETIC FIELD IS ASSOCIATED WITH THIS DISEASE?	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Kaune (Kaune, 1994a, 2002) found childhood cancer (including brain cancer) more associated with 180 Hz than 60 Hz. There was not a clear support for AC/DC resonance. (C2) Preston-Martin (Preston-Martin et al., 1996b) explored resonance with DC fields, time above 2 mG, and average size of the difference between consecutive measurements and found little or no evidence to support an effect from these metrics. (C3) Magnetic fields over water pipes in the Preston-Martin (Preston-Martin et al., 1996b) study were not associated with childhood brain cancer either. (C4) (Savitz et al., 1988) found no association with electric fields. (C5) Preston-Martin (Preston-Martin et al., 1996b) observed that peaks (the 90th percentile) during 24-hour measurements in the child's bedroom and "other" room studies showed ORs of 2-3 for the highest category of 4-22 mG. Those had wide confidence intervals.	(I1) Not enough evidence to focus on alternative metrics or aspects.

TABLE 10.4.2

EVIDENCE FOR THRESHOLD OR PLATEAU	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Any associations begin to appear at or above 3 mG. It is not clear if this is a threshold.	(I1) None.

TABLE 10.4.3

EVIDENCE FOR BIOLOGICAL WINDOWS OF VULNERABILITY	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) No evidentiary base.	(I1) None.

TABLE 10.4.4

CONSISTENT INDUCTION PERIOD OR REQUIRED DURATION OF EXPOSURE	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Birth and death address wire code were equally associated in Wertheimer's (Wertheimer & Leeper, 1979) study. (C2) Tynes (Tynes & Haldorsen, 1997) found larger (but imprecise) ORs with first year address rather than with diagnosis address. (C3) Swedish/Danish meta-analysis (Feychting et al., 1995) shows a larger imprecise association for year of diagnosis exposure than cumulative lifetime exposure.	(I1) Some suggestion of efficacy of recent exposure but the evidence is very weak.

TABLE 10.4.5

EMF COMPARED TO OTHER RISK FACTORS FOR THIS DISEASE	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Aside from genetic risk factors, there are few established risk factors for childhood brain cancer, and they do not convey high relative risks (Kuijten & Bunin, 1993). (C2) The relative size of the association may be relevant for risk communication but not for cost-benefit oriented policy.	(I1) None.

TABLE 10.4.6

RELATIVE RISK COMPARED TO THAT WHICH WOULD GENERATE 1/1000 OR 1/100,000 THEORETICAL LIFETIME RISK	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) With an annual incidence of a few cases per 100,000, 20 years of RR of 1.2 would accumulate an added risk above 1/100,000 and if real would be of regulatory concern. The degree of certainty about this association is quite low.	(I1) Could be of regulatory concern if real.

TABLE 10.4.7

EVIDENCE FOR RACIAL OR CLASS DIFFERENCES IN EXPOSURE OR VULNERABILITY	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) No evidentiary base.	(I1) None.

TABLE 10.4.8

ROOM FOR IMPROVEMENT IN QUALITY OR SIZE IN BEST EXISTING STUDIES	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) The study designs have been state of the art, just not very powerful from a statistical point of view because childhood brain cancer is even more rare than leukemia and high exposures are rare. (C2) The use of surrogate metrics for exposure tends to bias associations toward a null result, but is not an argument against causality.	(I1) It will be difficult to improve on the existing studies.

TABLE 10.4.9

NEW STUDIES IN PIPELINE AND ABILITY TO CHANGE ASSESSMENT	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) A large case-control study by Kabuto et al. is planned for Japan.	(I1) Could be influential regardless of results because of projected size and equivocal nature of existing evidence.

TABLE 10.4.10

HOW LIKELY IS IT THAT FURTHER STUDIES COULD RESOLVE CONTROVERSIES?	
COMMENT AND SUMMARY	**IMPACT ON POLICY**
(C1) Exposure assessments which would examine magnetic fields, electric fields, contact currents, and shocks in the residential environment, and which used various summary exposure metrics, might indicate potential confounding between these EMF aspects and metrics and could guide future epidemiology and laboratory research. (C2) Childhood brain cancer is quite rare and would not drive a cost-benefit oriented policy. It may be more productive to focus on other, more common diseases.	(I1) Not clear that further information on this condition would drive EMF policy.

10.5 CONCLUSIONS ON POLICY-RELEVANT SCIENTIFIC ISSUES

10.5.1 DOSE-RESPONSE ISSUES

The associations with EMFs are not clear for this disease, nor is there a sufficient evidentiary base to speculate about pathogenic aspects of the EMF mixture or summary exposure metrics, which might be more strongly associated. Similarly, there is insufficient evidentiary base to provide insight into induction period or shape of dose-response relationships. There is no evidentiary base to address the issue of unequal vulnerability or exposure.

10.5.2 RESEARCH POLICY

There is one large case-control study in the pipeline from Japan. Even if it implicates EMF as a cause of childhood brain cancer, it likely will leave questions about dose response, pathogenic aspects of EMF mixture, etc. If it is well conducted and is a null study it probably would put the childhood brain cancer issue to rest. The rarity of this disease means that it would not drive a cost-benefit oriented policy and makes it difficult to conduct studies. This may not be a priority area for further research. The results of the Japanese study may conceivably alter this conclusion.